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In the United States, the practices and opinions of healthcare providers regarding eating on dialysis are unknown. The purpose of this study was to understand healthcare provider opinions and in-center hemodialysis patient practices regarding eating while at the dialysis center. In June 2011, over 1200 registered dietitians within a large dialysis organization in the US were surveyed on current practices and opinions of patient food consumption during dialysis treatment using an online survey.

1223 of 1665 (73%) dialysis facilities responded to the food consumption survey.

n = 1222	Permitted n (%)	No Guidelines n (%)	Not allowed n (%)
Eating on dialysis	803 (66%)	67 (5%)	352 (29%)
Drinking on dialysis	907 (75%)	87 (7%)	228 (19%)

218 (18%) of the respondents stated that the facility practice for consuming nutritional supplements (eg., liquid nutritional supplements and/or protein bars) while on dialysis was different than the policy for consuming food while on dialysis. Interestingly, 1203 (98%) of the respondents stated consuming food before or after dialysis was allowed.

The top reasons for facility practices that allowed eating during dialysis were: prevention of hypoglycemia on dialysis, improved kcal intake on dialysis days, and the opportunity to provide counseling on food products currently chosen by the patient. The top reasons for facility practices not permitting eating during dialysis included: potential adverse events associated with hypotension, GI symptoms, choking, infection, pest control, and spills. Further analyses are warranted to determine whether there is a correlation between allowing patients to eat during dialysis treatment and an improvement in the nutritional status of the patients.

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TREATMENT OF CHRONIC KIDNEY DISEASE PATIENTS WITH A SUPPLEMENTED LOW PROTEIN DIET AND A SUPPLEMENTED VERY LOW PROTEIN DIET

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The primary results of the Modification of Diet in Renal Disease were inconclusive and did confuse a lot of physicians about the dietary approach to CKD management. The study design was flawed and thus compromised the results and conclusions. Re-analysis of the MDRD study however clearly showed the benefits of dietary protein restriction and also more importantly an additional benefit by ketoanalogue supplementation in delaying progression of CKD. Despite the obvious benefits of protein restriction, concern has been raised recently especially patients on very low dietary protein (very-low-protein diets; VLPDs), which could lead to deterioration in the nutritional status of CKD patients. To address this particular issue of whether a sVLPD diet induces malnutrition the present study has been taken up 132 adult patients with Stage 3 to Stage 5 (Predialysis) were initiated on a protein restricted ketoanalogue supplemented diet after informed consent and the necessary Institutional Ethics Committee approvals. Based on their affordability, 92 patients randomly were assigned to the sLPD group whereby they received 0.6 G/Kg BW of dietary proteins supplemented by ketoanalogues at a dosage of one tablet per 10 Kg body weight. 40 patients received 0.3 G/Kg BW supplemented by ketoanalogues at a dose of one tablet per 5 Kg body weight. Renolog[®] tablets manufactured by La Renon Healthcare Ltd, Ahmedabad, India were prescribed as the ketoanalogue supplements. Renal, Metabolic, Nutritional parameters and Anthropometric analysis were done in both groups at the start of the study and at the end of 6 months of follow up. The mean blood urea in the SLPD group showed a decrease from 81.17 ± 0.00 mg/dl to 74.45 ± 30.75 mg/dl ($p < 0.05$) and in the SVLPD from 78.35 ± 0.00 mg/dl to 66.50 ± 34.50 mg/dl ($p > 0.05$) at the end of six months indicating an improvement in renal function. The serum creatinine also showed a decrease from 3.52 ± 0.00 mg/dl to 3.30 ± 1.63 mg/dl ($p > 0.05$) in the SLPD group and a decrease from 3.74 ± 0.00 mg/dl to 3.55 ± 1.67 mg/dl ($p > 0.05$) in the SVLPD group though not statistically significant. The eGFR showed improvement from 26.76 ± 0.00 ml/min to 30.75 ± 17.31 ml/min ($p < 0.05$) at end of six

months in the SLPD group and an increase from 23.62 ± 0.00 ml/min to 26.35 ± 10.58 ml/min ($p > 0.05$) in the SVLPD group. Serum albumin increased from 3.85 ± 0.00 gm/dl to 4.00 ± 0.56 gm/dl ($p < 0.05$) in the SLPD group and an increase from 4.03 ± 0.00 gm/dl to 4.07 ± 0.47 gm/dl in the SVLPD group indicating an improvement in nutrition in SVLPD group ($p > 0.05$). Blood hemoglobin increased from 11.18 ± 2.02 gm/dl to 11.48 ± 2.14 gm/dl in the SLPD group and an increase from 11.35 ± 0.96 gm/dl to 13.22 ± 1.03 gm/dl in the SVLPD group. Combined analysis of both the study groups together (SLPD + SVLPD) showed a statistically significant decrease in blood urea from 80.32 ± 0.0 mg/dl to 73.70 ± 31.81 mg/dl ($p < 0.05$), decrease in serum creatinine from 3.59 ± 0.0 mg/dl to 3.38 ± 1.64 mg/dl ($p > 0.05$) and increase in eGFR from 25.82 ± 0.0 ml/min to 29.42 ± 15.67 ml/min ($p < 0.05$) at the end of six months of therapy which was statistically significant indicating an improvement in renal function. The serum albumin increased from 3.91 ± 0.0 gm/dl to 4.02 ± 0.53 gm/dl ($p < 0.05$) indicating a statistically significant improvement in nutrition. The protein restricted ketoanalogue treated patients in this large series showed significant improvement in their renal function, metabolic status and their extensively investigated nutritional parameters over a period of 6 months. The key here is to use the right dosage of the ketoanalogue supplements in addition to ensuring strict compliance of dietary restrictions. It is strongly recommended that ketoanalogues be routinely used in the conservative management of CKD.

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INDOXYL SULFATE UPREGULATES RENAL EXPRESSION OF ICAM-1 VIA PRODUCTION OF ROS AND ACTIVATION OF NF-κB AND P53 IN PROXIMAL TUBULAR CELLS

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Intercellular adhesion molecule 1 (ICAM-1) plays an important role in adhesion of monocytes/macrophages to injured tubulointerstitial tissue. The present study examined whether indoxyl sulfate, a uremic toxin, regulates renal expression of ICAM-1. The effect of indoxyl sulfate on expression of ICAM-1 was determined using human proximal tubular cells (HK-2 cells) and following animals: (1) Dahl salt-resistant normotensive rats (DN), (2) Dahl salt-resistant normotensive indoxyl sulfate-administered rats (DN+IS), (3) Dahl salt-sensitive hypertensive rats (DH), and (4) Dahl salt-sensitive hypertensive indoxyl sulfate-administered rats (DH+IS). DN+IS, DH, and DH+IS rats showed significantly increased mRNA expression of ICAM-1 in the kidneys compared with DN rats. DH+IS rats showed significantly increased mRNA expression of ICAM-1 in the kidneys compared with DH rats. Indoxyl sulfate upregulated mRNA expression of ICAM-1 in HK-2 cells. Inhibitors of NADPH oxidase, NF-κB and p53 suppressed indoxyl sulfate-induced mRNA expression of ICAM-1 in HK-2 cells.

In conclusion, indoxyl sulfate upregulates renal expression of ICAM-1 through production of ROS and activation of NF-κB and p53 in proximal tubular cells. Thus, accumulation of indoxyl sulfate in chronic kidney disease might be involved in the pathogenesis of tubulointerstitial injury through induction of ICAM-1 in the kidneys.

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ORAL ACTIVE VITAMIN D THERAPY AS A POTENTIAL CHEMOPREVENTION AGAINST POST-TRANSPLANT MALIGNANCY

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Post-transplant malignancy (PTM) is a limiting factor for both patient and allograft survival in kidney transplant recipients (KTRs). We hypothesized that active vitamin D compounds (AVDs) could reduce the development of PTM in KTRs and evaluated the effects of AVD therapy in a prospective cohort of ambulatory KTRs in a Japanese single center. We used a propensity score (PS) of having received AVDs estimated by 25 clinically relevant factors to adjust for these confounders.

Among 218 participants, the mean age was 49.4 (SD, 12.1) years, 63.3% were male, the median time since transplantation was 11.2 (interquartile range [IQR], 5.2–17.1) years, and 42.2% had been treated with AVDs at